IN THE CLAIMS

Please amend the claims as follows.

 (Currently Amended) A rat with impaired performance in memory and learning and having a neurologie disease hyperphosphorylated tau, amyloid plaques or neurofibrillary tangles induced by the process of:

perfusing [[the]] a rat without impaired performance in memory and learning with a pharmacologically effective amount of a combination of an Aβ compound, at least one prooxidative compound, and at least one anti-oxidant inhibitor, to yield a rat with wherein the perfusion produces impaired performance of the rodent in memory and learning tests and induces abnormal neuropathology in a brain of the rodent, wherein said impaired performance and abnormal neuropathology are in comparison with control non-human rats rodents, [[and]] wherein the anti-oxidant inhibitor inhibits glutathione synthesis, and wherein the abnormal neuropathology includes hyperphosphorylated tau, amyloid plaques or neurofibrillary tangles.

- (Currently Amended) The <u>impaired rat rodent</u> of claim 1, wherein the Aβ compound comprises Aβ₄₂.
- 3. (Currently Amended) The <u>impaired rat</u> redent of claim 1, wherein the $A\beta$ compound comprises a peptide fragment of $A\beta_{42}$.
- 4. (Currently Amended) The <u>impaired rat rodent</u> of claim 3, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{1-40}$ or $A\beta_{24-35}$.
- (Withdrawn) The non-human animal of claim 1, wherein the Aβ compound comprises a
 peptidomimetic that mimicks Aβ₄₂.

- 6. (Currently Amended) The <u>impaired rat rodent</u> of claim 1, wherein the at least one prooxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.
- (Currently Amended) The <u>impaired rat rodent</u> of claim 1, wherein the at least one prooxidative compound comprises ferrous sulfate.
- (Currently Amended) The <u>impaired rat rodent</u> of claim 1, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.
- (Currently Amended) The <u>impaired rat rodent</u> of claim 1, wherein the process further comprises perfusing the <u>nonimpaired rat non-human animal</u> with an effective amount of a phosphatase inhibitor.
- 10. (Currently Amended) The <u>impaired rat rodent</u> of claim 9, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerate, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.
- (Currently Amended) The <u>impaired rat redent</u> of claim 9, wherein the phophatase phosphatase inhibitor comprises okadaic acid.
- 12. (Currently Amended) The <u>impaired rat redent</u> of claim 1, wherein the process further comprises perfusing the <u>nonimpaired rat non-human animal</u> with an effective amount of a proinflammatory compound.
- (Currently Amended) The <u>impaired rat rodent</u> of claim 12, wherein the pro-inflammatory compound is selected from the group consisting of TNF-α, IL-6, and IL-1b.

- (Currently Amended) The <u>impaired rat</u> rodent of claim 12, wherein the pro-inflammatory compound comprises TNF-α.
- 15. (Currently Amended) A method for inducing <u>hyperphosphorylated tau</u>, <u>amyloid plaques or</u> neurofibrillary tangles a <u>neurologie disease</u> in a rat, comprising:

perfusing [[the]] a rat with a pharmacologically effective amount of a combination of an $A\beta$ compound, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor that inhibits glutathione synthesis, wherein the perfusion results in the <u>rat redent</u> having hyperphosphorylated tau, amyloid plaques or neurofibrillary tangles.

- 16. (Original) The method of claim 15, wherein the A β compound comprises A β_{42} .
- 17. (Original) The method of claim 15, wherein the A β compound comprises a peptide fragment of A β ₄₂.
- 18. (Original) The method of claim 17, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{140}$ or $A\beta_{24:35}$.
- (Withdrawn) The method of claim 15, wherein the Aβ compound comprises a peptidomimetic that mimicks Aβ₄₂.
- 20. (Original) The method of claim 15, wherein the at least one pro-oxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.
- 21. (Original) The method of claim 15, wherein the at least one pro-oxidative compound comprises ferrous sulfate.

Filing Date: March 11, 2004
Title: ANIMAL MODEL SIMULATING NEUROLOGIC DISEASE

22. (Original) The method of claim 15, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.

- 23. (Currently Amended) The method of [[Claim]] claim 15, further comprising perfusing the rat non-human animal with an effective amount of a phosphatase inhibitor.
- 24. (Original) The method of claim 23, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerte, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.
- 25. (Original) The method of claim 23, wherein the phophatase inhibitor comprises okadaic acid.
- 26. (Currently Amended) The method of claim 15, further comprising perfusing the <u>rat</u> non-human animal with an effective amount of a pro-inflammatory compound.
- 27. (Original) The method of claim 27, wherein the pro-inflammatory compound is selected from the group consisting of TNF- α , IL-6, and IL-1b.
- 28. (Original) The method of claim 27, wherein the pro-inflammatory compound comprises TNF- α .

29-32. (Canceled)